## CLAIMS

We claim:

1. A method of detecting ivermectin sensitivity in a subject, comprising determining whether a gene-truncation mutation in a *mdr1*-encoding sequence of the subject or a truncated P-pg is present in the subject, wherein presence of the gene-truncation mutation or truncation of P-gp indicates that the subject is sensitive to ivermectin.

2. The method of claim 1, wherein the subject is a canine.

3. The method of claim 1, wherein the gene truncation mutation is a deletion of four base pairs at about residue 294-297 of SEQ ID NO: 1.

4. The method of claim 1, wherein the method is used to evaluate whether the subject can be treated safely with ivermectin or another drug that can be excluded from a cell or an organ by P-gp.

5. The method of claim 4, wherein the method is used to evaluate whether the subject can be treated safely with ivermectin or another drug that can be excluded from the brain by P-gp.

6. The method of claim 1, further comprising determining whether the subject is homozygous or heterozygous for the gene-truncation mutation.

7. The method of claim 1, wherein determining whether a gene-truncation mutation is present in the subject comprises subjecting DNA or RNA from the subject to amplification using oligonucleotide primers.

8. The method of claim 6, comprising an oligonucleotide ligation assay.

9. The method of claim 1, comprising:

obtaining a test sample of DNA containing a *mdr1* sequence of the subject; and

determining whether the *mdr1* sequence of the subject has the gene-truncation mutation in the *mdr1* sequence, wherein the presence of the mutation indicates sensitivity of the subject to ivermectin.

10. The method of claim 9, wherein determining whether the *mdr1* sequence of the subject has the mutation comprises using restriction digestion, probe hybridization, nucleic acid amplification, or nucleotide sequencing.

11. The method of claim 1, comprising:

obtaining from the subject a test sample of DNA comprising an *mdr1* sequence;

contacting the test sample with at least one nucleic acid probe for an *mdr1* gene truncation mutation that is associated with ivermectin sensitivity, to form a hybridization sample;

maintaining the hybridization sample under conditions sufficient for specific hybridization of the *mdr1* sequence with the nucleic acid probe; and

detecting whether the *mdr1* sequence specifically hybridizes with the nucleic acid probe, wherein specific hybridization of the *mdr1* sequence with the nucleic acid probe indicates ivermectin sensitivity of the subject.

12. The method of claim 10, wherein the probe is present on a substrate.

13. The method of claim 12, wherein the substrate is a nucleotide array.

14. The method of claim 1, comprising determining whether truncated P-gp is present in a sample from the subject.

15. The method of claim 14, comprising reacting at least one P-gp molecule contained in the sample from the subject with a P-gp-specific binding agent to form a P-gp:agent complex.

16. The method of claim 15, wherein the binding agent is an antibody.

17. The method of claim 15, further comprising detecting the P-gp:agent complex.

18. The method of claim 17, wherein the complexes are detected by Western blot assay.

19. The method of claim 17, wherein the complexes are detected by ELISA.

20. A method of making a treatment decision for a subject, comprising determining whether a gene-truncation mutation in a *mdr1*-encoding sequence of the subject or a truncated P-pg is present in the subject, wherein presence of the gene-truncation mutation or truncation of P-gp influences the treatment decision.

21. The method of claim 20, wherein the treatment decision relates to a decision of whether to provide the subject with ivermectin or another drug that can be excluded from a cell or an organ by P-gp.

22. A kit for use in diagnosing or detecting ivermectin sensitivity in a subject, comprising a probe that specifically hybridizes to an *mdr1* gene-truncation mutation associated with ivermectin sensitivity.

23. The kit of claim 22, wherein the probe specifically hybridizes to an *mdr1* gene-truncation mutation at or about residue 294-297 of SEQ ID NO: 1.

24. A kit for use in diagnosing ivermectin sensitivity in a subject, comprising a P-gp-specific binding agent.

25. The kit of claim 24, wherein the binding agent is an antibody.

26. The kit of claim 24, wherein the binding agent is capable of specifically binding to truncated P-gp protein.

27. An oligonucleotide that specifically hybridizes to a canine *mdr1* gene-truncation mutation.

28. The oligonucleotide according to claim 27, wherein the oligonucleotide hybridizes to an *mdr1* gene-truncation mutation at residue 294-297.

29. A method of determining a P-gp influenced biological effect of a compound on a canine cellular system, comprising:

contacting a canine cell with the compound, wherein the cell has a truncation mutation in its *mdr1* gene, and

comparing a characteristic of the canine cell contacted with the compound with the characteristic of a similar canine cell not contacted with the compound, wherein a difference in the characteristic between the two cells is indicative of the P-gp influenced biological effect.

30. The method of claim 29, wherein the characteristic of the cell is genetic, physiological, chemical, or morphological.

31. The method of claim 29, wherein the canine cell is a Collie cell.

32. The method of claim 29, where in the truncation mutation in the *mdr1* gene is a mutation at residue 294-297.

33. The method of claim 29, wherein contacting the canine cell with the compound occurs *in vivo* in the native environment of the canine cell.

34. The method of claim 29, wherein the biological effect is absorption or distribution of a drug or compound.

35. The method of claim 29, wherein the canine cell is a gastrointestinal tissue cell, a renal tissue cell, a brain capillary endothelial cell, or a liver tissue cell.

36. The method of claim 29, wherein the canine cell is a neoplastic cell.

37. An animal model useful for studying a P-gp influenced biological effect of a compound, comprising a Collie identified as being homozygous or heterozygous for a truncation mutation in the *mdr1* gene.

38. The animal model of claim 37, wherein the truncation mutation is a mutation at residue 294-297.

39. The method of claim 29, wherein the compound is a known or potential neurokinin receptor antagonist, anti-emetic agent, beta-adrenergic receptor antagonist, antiinfective agent, antiepileptic agent, antineoplastic agent, analgesic agent, anti-psychotic agent, or anti-depressive agent.

40. The method of claim 39, wherein the compound is an anti-infective agent and the anti-infective agent is an antiviral agent.

41. The method of claim 37, wherein the compound is a known or potential neurokinin receptor antagonist, anti-emetic agent, beta-adrenergic receptor antagonist, antiinfective agent, antiepileptic agent, antineoplastic agent, analgesic agent, anti-psychotic agent, or anti-depressive agent.

42. The method of claim 41, wherein the compound is an anti-infective agent and the anti-infective agent is an antiviral agent.